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PHYTOPATHOLOGY

VOLUME XI NUMBER 9

SEPTEMBER, 1921

MOSAIC DISEASE OF SUGAR BEETS

W. W. Robbins

With Eight Figures in the Text

INTRODUCTION

The following account of mosaic of sugar beet is based upon field observations and experimentation in northern Colorado and western Nebraska. It has been thought well to place the data and conclusions obtained to date in published form, in view of the rather exceptional interest among plant pathologists at this time in the mosaic diseases.

Scattered cases of sugar beet mosaic were observed in beet fields of the irrigated sections of northern Colorado in 1917, more in 1918 and 1919, and an increasing number in 1920. At present it appears to be confined chiefly to seed beets and stecklings[1] and to fields of commercial beets adjacent to diseased seed beets or stecklings. Field observations in northern Colorado, eastern Colorado and western Nebraska in 1920 showed no cases of transmissible mosaic on commercial beets which were more than one to one and one-half miles from possible sources of infection.

It is the practice in the beet seed growing sections to raise stecklings and seed bearing plants on the same farm. The obvious reason for this is to save labor in the spring in the hauling of roots from the silo to the point where they are to be set out. Consequently, steckling plants and seed-bearing plants are frequently in close proximity. This undoubtedly accounts for the greater prevalence of mosaic on stecklings than on commercial beets, except those near diseased seed beets, for diseased seed-bearing plants are the chief sources of infection.

[1] Beets held over winter for the production of seed the following season are called "stecklings." The seed from which stecklings are grown is planted at the same time and in the same manner as commercial beet seed. Commercial beets are thinned by hand, leaving one beet every 10 to 14 inches. Stecklings, however, are not thinned at all, or only by harrowing crosswise the rows, thus leaving three to five beets, or more, per foot. Prior to digging in the fall, the tops are mowed off; the stecklings are then siloed in earth trenches.

As far as the writer can determine, mosaic of sugar beets has not attracted attention in any other of the sugar beet growing sections of this country or of Europe. Lind (4) reports mosaic on garden beets in Denmark, and a few other places, chiefly southern Sweden, the north of France, and Berlin, but specifically mentions that the disease is never found on sugar beets.

The mosaic of sugar beet described herein is distinct from curly-top of sugar beet, a disease so prevalent in Utah and California.

The economic importance of curly-top has stimulated extended investigation. The most important facts relating to curly-top may be found in publications by Linhart (5), Townsend (9), Ball (2), Shaw (7), Bunzel (3), and Smith and Boncquet (8).

In northern Colorado and western Nebraska, where mosaic of sugar beet is most prevalent, true curly-top rarely occurs. One may find in an entire season's field work only a few isolated cases of diseased beets which even suggest curly-top.

The only symptoms of mosaic and curly-top in common are curling and crinkling of the leaves. The inward and retracted type of leaf curl in curly-top is seldom if ever found in mosaic-affected beets. The vein distortion of curly-top is far more pronounced than, and quite different in character from, that in mosaic. Vein distortion does not occur in primary-infected mosaic leaves. Moreover, in no cases does vein distortion appear as knot-like swellings, so typical on leaves of curly-top. Nipple-like protuberances, so characteristic of curly-top leaves, are never associated with mosaic. In mosaic plants there is no shortening and bowing inward of the petioles as in the leaves of plants with curly-top. The crown of mosaic plants remains normal, and the roots never throw out dense masses of rootlets; furthermore, there is no apparent stunting of first-year beets, and seldom of second-year beets. The brittleness of mosaic leaves is confined to secondarily infected plants, and has not been observed on plants with primary infection. This is unlike curly-top. The outstanding symptom of beet mosaic is a pronounced mottling of the leaves, which symptom is not evident in curly-top.

There is another sugar beet disease scattered throughout commercial fields of the territory studied which has some mosaic characters, but which the writer believes to be distinct from true transmissible mosaic. The plants are distinguished by their asymmetry. One-half of the plant, including both top and root, is dwarfed; the other half is apparently normal. The affected leaves are small, thick, curled, twisted and the mesophyll tissue bordering the veins of the leaf is pallid. The root shows eccentric growth, and seldom attains normal size. All attempts to transmit this disease by means of aphids have met with failure.

Under similar conditions, however, true mosaic is readily transmitted by aphids. Moreover, in the scope of our observations, diseased plants of the asymmetrical type are not centers of infection in the field, whereas plants with true mosaic are centers of infection.

No definite data can yet be presented as to the effect of mosaic on quality and quantity of seed produced, or upon the yield and quality of roots. However, here and there in seed beet fields are plants so badly diseased, with leaves so heavily mottled, crumpled, twisted and contorted, that the yield of seed is reduced to a very small amount.

SYMPTOMS

Mottling

Mottling of the leaves is the characteristic symptom of sugar beet mosaic. This mottling is produced by the occurrence of light green areas on the foliage. There is much variation in the degree of paleness in the lighter green patches; in some instances these are barely discernible by reflected light, whereas if the leaf is observed by transmitted light the spots or areas are plainly visible. Frequently, the paler areas are very sharply delimited from the bordering normal green of the mesophyll; and usually in such instances the paler portion takes the form of a ring surrounding darker tissue.

The light green areas also vary widely in shape. The leaf may appear finely speckled, the areas being very minute dots. This condition was observed chiefly on first-year beets grown in the greenhouse. Or, the areas may be elongate, circular, angular or irregular.

Any part of the leaf may be involved. A young leaf unfolding may be mottled throughout from base to tip; the pallid areas in such are usually very irregular, appearing as light green blotches. Again, a leaf may be mottled only at the base, the portion involved varying considerably in size. In a day or two, the mottling may spread from base to tip. Frequently, only one-half of a leaf shows mottling. The normal half may remain so throughout the season, or later become affected in a manner similar to its adjoining half. Seldom does one observe a leaf mottled only at the tip.

In the youngest leaves, the pallid, affected tissue may predominate, whereas in older leaves the normal dark green tissue usually predominates. Infected plants are seldom lighter in color than healthy individuals, and it is rarely possible to identify a diseased plant from a distance.

If one examines a plant in mid-season, the mosaic is most conspicuous on the young heart leaves. The pattern may be entirely masked or very much diffused on the older leaves. However, a mature plant may manifest mosaic symptoms on every leaf. This condition has been observed in both first-year and second-year beets.

There are instances of seed beets with distinct mottling extending upward to include not only the floral bracts but the perianth segments. However, no distortion of stamens and pistils has been observed.

Malformation

Malformation may or may not be associated with mottling. As a rule, however, a young leaf with marked mottling shows puckering and crinkling of the mesophyll tissue.

A very characteristic malformation, chiefly on seed beets grown under greenhouse conditions, is a bending back of the leaf near the tip. About

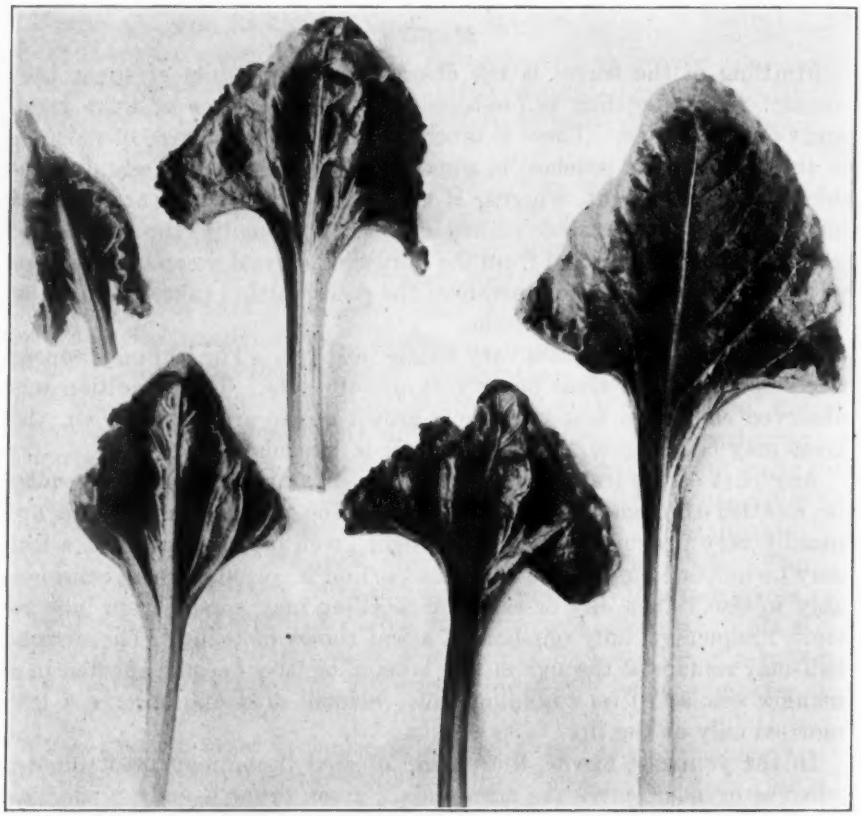

FIG. 1. LEAF MALFORMATIONS ASSOCIATED WITH MOSAIC OF SUGAR BEET
The curling at the tip is preceded by necrosis in the midrib back of the leaf tip.

one-third of the way back from the leaf tip, marginal growth apparently ceases earliest, as the most pronounced curling is at this point on both margins. The main vein from which lateral veins arise, leading to these marginal points, becomes affected first. This affection is first

manifest by a crinkling and apparent shortening of the vein, as though the vascular strands were being contracted lengthwise. Later, the vein so affected becomes unevenly discolored, and later totally darkened and shrivelled. (Figs. 1 and 2.) The cessation of growth at the margins

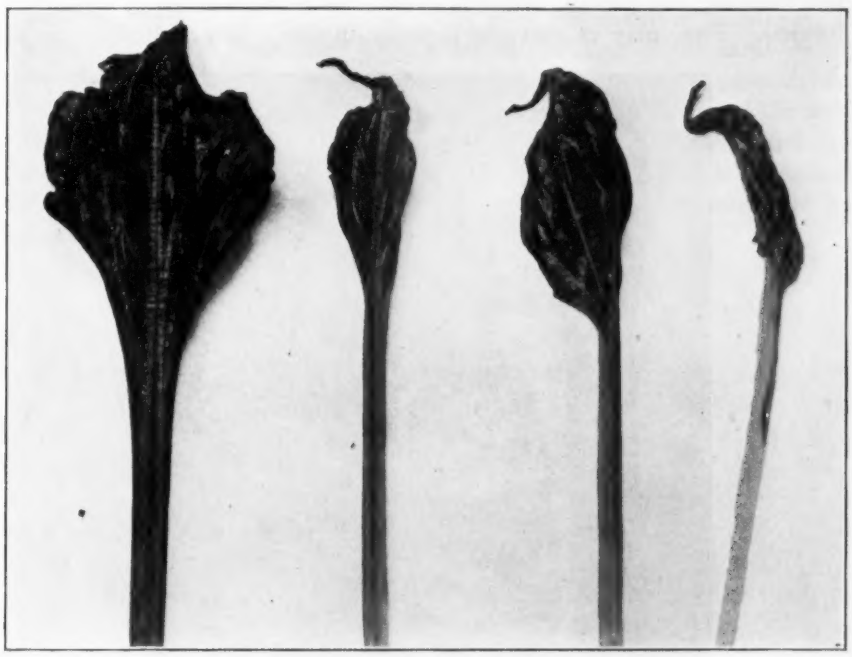

FIG. 2. LEAF MALFORMATIONS ASSOCIATED WITH MOSAIC OF SUGAR BEET
More advanced stages than those shown in Figure 1.

is preceded and caused by some disturbance in the functioning of the vascular tissue which is tributary to any particular marginal zone.

This folding back of the leaf tip, sometimes to form a pocket, the curling and crinkling of the leaf margin and tip, and frequently death of the tip later, are very striking and constant symptoms associated with mottling and greenhouse-grown seed beets. The same symptoms have been observed on seed beets in the field, but the proportion showing them is far less than that of plants in the greenhouse. The crinkling and puckering in extreme cases may take the form of conspicuous, bladdery areas over the leaf surface.

The first leaves produced from a mother beet carrying mosaic, are usually yellowish green, mottled, much curled, crumpled and crinkled and abnormally thick and brittle. This crinkling and puckering often occur in young leaves from a normal mother beet, but in a leaf showing

both malformation and mottling, a degree of crinkling and puckering approaching abnormality is attained. Leaves affected as above may partially outgrow these symptoms, and become quite normal as far as can be ascertained by the eye. In most instances, however, they do not recover, but remain mottled, thick, brittle and more or less malformed. They may or may not increase in size. (Fig. 3.)

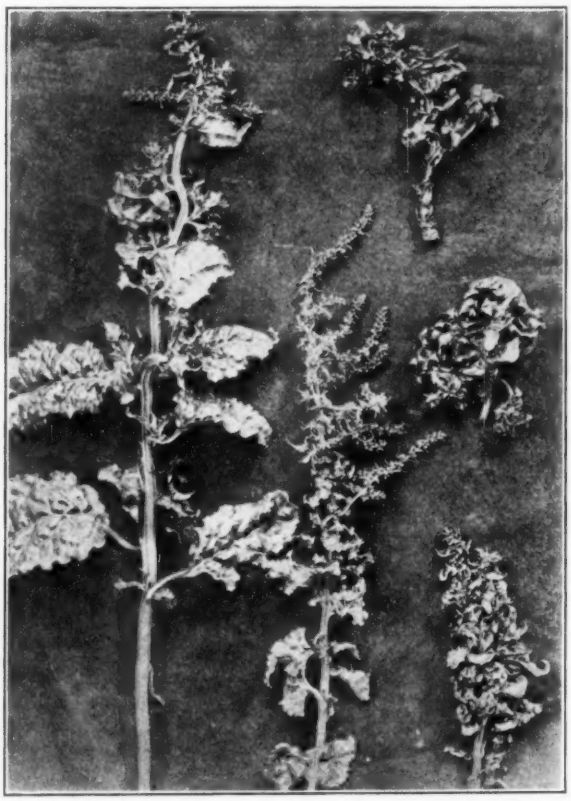

FIG. 3. MOSAIC OF SUGAR BEETS
Heavily mottled and malformed leaves and floral bracts.

The environmental conditions under which the plants grow appear to be associated with the expression of mosaic. The mottled phase without malformations is predominant under field conditions, both as concerns first-year and second-year growth. Seed plants in the greenhouse usually develop marked malformations in leaf growth.

PATHOLOGICAL ANATOMY OF DISEASED PARTS

In a cross-section of the mid-rib in that portion of the leaf of a seed plant showing curling and very marked distortions, the vascular area

and tissue adjoining show characteristic symptoms. (Fig. 4.) There is a darkening of the vascular tissue and cells adjacent thereto; many of the phloem elements are filled with a yellowish or reddish brown substance; the walls of the phloem cells are darker in color and thicker than normal; wood fibers are for the most part not involved; however, in advanced stages, these may have a deposit in them of the same brown material found in other cells of the vascular region. In the parenchyma cells bordering the vascular strands, there may be deposits of the substance mentioned above; and also in the intercellular spaces such deposits are occasionally found. This pathological condition resembles that of phloem-necrosis observed in potato plants, as described by Quanjer, (6) and Artschwager, (1).

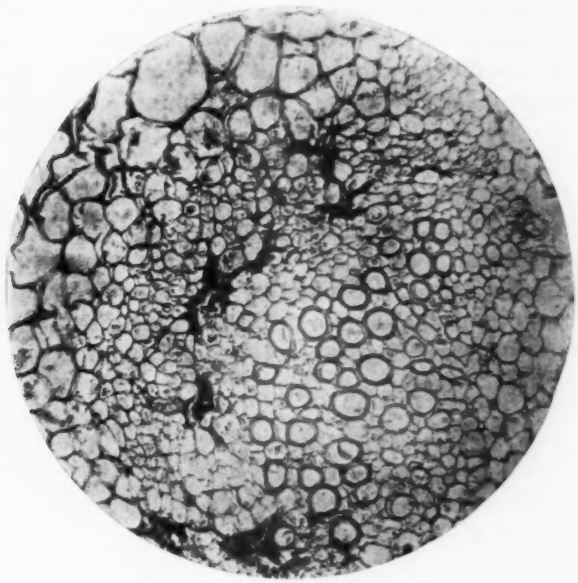

FIG. 4. NECROTIC PHLOEM IN VASCULAR BUNDLES OF MIDRIB OF LEAF SHOWING MALFORMATIONS ILLUSTRATED IN FIGURES 1 AND 2.

As would be expected, in leaves which have necrotic phloem tissue in the veins the transportation of carbohydrates, manufactured beyond the diseased portion, is hindered. This is evidenced by abnormal starch accumulations. The normal, fresh, green beet leaf contains no starch, except late in the afternoon of a bright, sunny day, at which time very small granules may be detected in the plastids. Primarily infected beet leaves are similar to normal ones as to the starch accumulation within them. On the other hand, thick brittle, heavily mottled leaves of secondary diseased plants (seed beets) usually show an abundance of starch within them, when tested at any time during the day, although

more in the afternoon than in the forenoon. That portion of a leaf beyond the parts of the veins which have necrotic phloem, shows an extraordinary accumulation of starch.

DEVELOPMENT OF DISEASE

The appearance of external symptoms comes on very abruptly as a rule. Individuals apparently free from mosaic one day, will manifest unmistakable symptoms the following day. Usually the first mottling shows up on the heart leaves, and in 5 to 7 days, the mottling has spread, generally, to include its maximum area.

The mosaic pattern may disappear entirely or later be demonstrated with difficulty in first-year, as well as in second-year sugar beets.

Cases of apparent recovery from sugar beet mosaic have been observed. In three plots of seed beets containing a total of 684 individuals, 26.7 per cent of all those which showed mosaic at the beginning of the season or at some date throughout the season were apparently free from the disease at harvest date. The emphasis here needs be placed upon "apparently", for it is altogether likely that such individuals are still sources of infection.

The fact that a plant has outgrown its symptoms is no evidence that it has recovered from the disease. This view is further strengthened by experiments with stecklings. Beets which were apparently free from mosaic when taken in September from plots containing diseased individuals were topped, and set out in the greenhouse. The first leaves which developed showed characteristic mosaic symptoms.

PERIOD OF INCUBATION

The cage experiments described below establish an incubation period on seed beets under greenhouse conditions of approximately 24 days; and on young seedling plants bearing eight to ten leaves growing under similar conditions, of 12 to 18 days.

There was in 1920 a gradual increase in the amount of mosaic in the experimental plots through the season, as figure 8 graphically shows, but the most rapid increase came after August 21. It is possible that this phenomenon is related to the appearance and migration of insect broods.

TRANSMISSION OF THE DISEASE

Field Studies

There is abundant evidence that the mosaic of sugar beet is carried from diseased to healthy individuals.

A tract of land south of Longmont, Colorado, was planted April 7–9, 1920, to mother beets grown on the experimental farm of the Great Western Sugar Company in 1919. There were 11,122 individual seed plants on this tract. On May 10, approximately 55 per cent of all

plants had unmistakable mosaic symptoms. On this date, the roots had produced a cluster of small leaves. By July 6, 99.3 per cent of all plants showed the mosaic disease. (Fig. 5.)

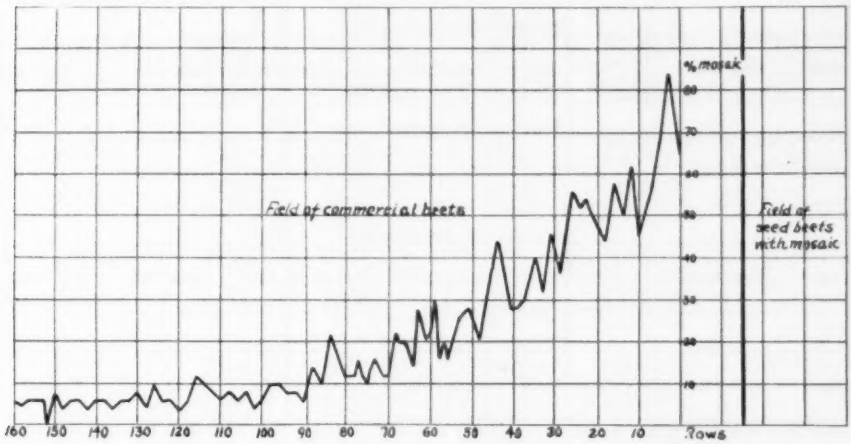

FIG. 5. SPREAD OF SUGAR BEET MOSAIC IN THE FIELD

The field directly north of the tract growing the diseased seed beets, was planted to commercial beets on April 15. On July 24, the percentage of mosaic in this field was determined. In doing this the percentage of mosaic was obtained for each row up to number 84, and from there on for every other row up to row number 180. Row 1 (Fig. 5) is nearest the field of diseased seed beets, and row 160, farthest from it. If insects carry mosaic from affected seed beets to commercial beets, it is altogether probable that the rows closest to the diseased beets would have the largest percentage of individuals with mosaic, and that there would be a decrease in the percentage across the field. That this is the case may be seen from figure 5.

On the experimental farm of the Great Western Sugar Co., Longmont, Colorado, there are, in one of its rotation experiments, 21 plots of approximately one-twentieth acre each. In each series there are 7 plots, which are in triplicate. The cropping on these plots is shown in figure 6.

It will be noted that three of the plots, 3, 3′ and 3″, were planted to mother beets in 1920. The mothers were set out from March 31 to April 6. On May 17, 95.7 per cent of the plants on plot 3, 93.5 per cent of those on plot 3′, and 97.9 per cent of those on plot 3″ showed distinct mottling. At this time, the leaves were very small, and in a dense cluster on the crown. Plots 1, 4, 5 and 7 of each series were seeded to commercial beets on May 17. The young plants were emerging on May 25.

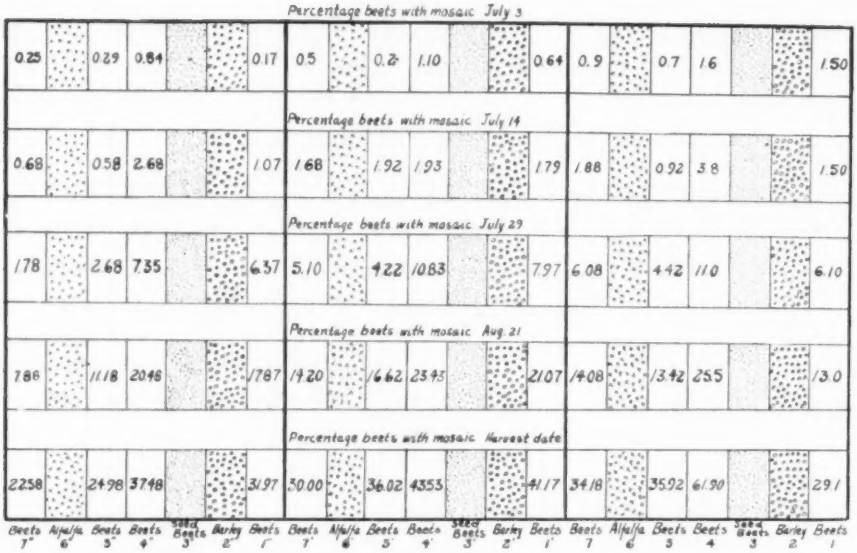
Percentage beets with mosaic July 3
0.25 0.29 0.84 0.17
0.5 0.2 1.10 0.64
0.9 0.7 1.6 1.50
Percentage beets with mosaic July 14
0.68 0.58 2.68 1.07
1.68 1.92 1.93 1.79
1.88 0.92 3.8 1.50
Percentage beets with mosaic July 29
1.78 2.68 7.35 6.37
5.10 4.22 10.83 7.97
6.08 4.42 11.0 6.10
Percentage beets with mosaic Aug. 21
7.86 11.18 20.48 17.87
19.20 16.62 23.43 21.07
14.08 13.42 25.5 13.0
Percentage beets with mosaic Harvest date
22.58 24.98 37.48 31.97
30.00 36.02 43.53 41.17
34.18 35.92 61.90 29.1
Beets 7' Alfalfa 6' Beets 5' Beets 4' Seed Beets 3' Barley 2' Beets 1'
Beets 7' Alfalfa 6' Beets 5' Beets 4' Seed Beets 3' Barley 2' Beets 1'
Beets 7 Alfalfa 6' Beets 5 Beets 4 Seed Beets 3 Barley 2' Beets 1

FIG. 6. SPREAD OF SUGAR BEET MOSAIC ON ROTATION PLOTS.
Plots 3, 3′ and 3″ containing diseased seed beets are sources of infection. Note that the plots of commercial beets nearest to affected seed beets have the largest percentage of diseased individuals: also note increase in any one plot as the season advances.

These plots were examined frequently to detect the first evidence of mosaic. It was suspected that the disease would be carried from the adjacent affected seed beet plots. The first cases of mosaic on the seedling plots were on July 3, as follows:

TABLE 1

Mosaic on sugar beet seedlings, July 3

PLOT	MOSAIC NO. PLANTS	MOSAIC PERCENTAGE OF PLANTS
1	31	1.5
4	36	1.6
5	12	0.7
7	21	0.9
1′	10	0.64
4′	20	1.10
5′	4	0.20
7′	9	0.50
1″	3	0.17
4″	16	0.84
5″	5	0.29
7′	4	0.25

TABLE 2

Summary of each plot of commercial beets:

Rotation 1, showing total numbers of beets with mosaic on certain dates

PLOT	JULY 3		JULY 10		JULY 14		JULY 22		JULY 29		AUG. 6		AUG. 21		AUG. 28		SEPT. 13–24		WITHOUT MOSAIC
	NEW:	TOTAL	NEW:	TOTAL	NEW:	TOTAL	NEW:	TOTAL	NEW:	TOTAL	NEW:	TOTAL	NEW:	TOTAL	NEW:	TOTAL	NEW:	TOTAL	
1	31	31	0	31	0	31	47	78	44	122	46	168	90	258	133	391	182	573	1384
4	36	36	24	60	24	84	86	170	69	239	107	346	205	551	524	1075	261	1336	812
5	12	12	3	15	1	16	35	51	26	77	50	127	106	233	172	405	216	621	1098
7	21	21	8	29	13	42	50	92	40	132	57	189	114	303	232	535	196	731	1390
1′	10	10	8	18	10	28	52	80	43	123	61	184	144	328			315	643	920
4′	20	20	10	30	25	55	78	133	91	224	77	301	161	462			379	841	1042
5′	4	4	7	11	21	32	22	54	33	87	62	149	140	289			316	505	1118
7′	9	9	4	13	17	30	34	64	28	92	31	123	132	255			281	536	1235
1″	3	3	0	3	16	19	66	85	26	111	52	163	144	307			230	537	1157
4″	16	16	6	22	28	50	53	103	37	140	42	182	208	390			324	714	1184
5″	5	5	4	9	1	10	16	26	21	47	30	77	117	194			239	433	1287
7″	4	4	0	4	7	11	8	19	10	29	21	50	77	127			235	462	1137

Note—On account of irrigation and rain it was impossible to make observations on Plots 1′ to 7′, on August 28.

It will be noted from the above that the largest percentage of diseased plants occurs on plots 4, 4′ and 4″ which are adjacent to the affected seed beets, plots 3, 3′ and 3″. At all times throughout the season, and at harvest time, the plots closest to the diseased seed beets showed the highest percentage of mosaic.

This fact is shown in tables 2 and 3, and in figures 6 and 7.

If we follow the progress of mosaic by rows in plots 4, 4′ and 4″, which border the diseased seed beet plots, we again see that mosaic spreads from the diseased plants as a center. This fact is brought out in the table 3 and figures 6 and 7. In plots 4, 4′ and 4″, row 1 in each case is nearest the affected seed beets, of plots 3, 3′ and 3″.

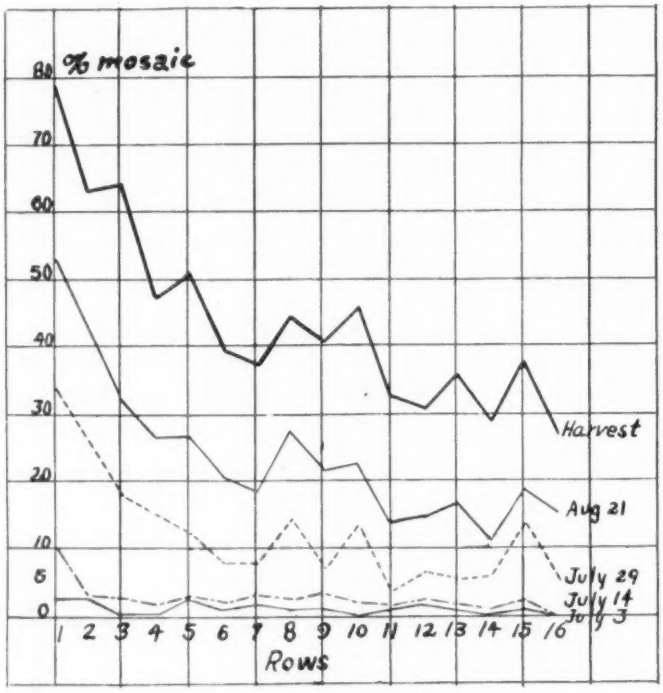

FIG. 7. PERCENTAGE OF MOSAIC BY ROWS ON FIVE SUCCESSIVE DATES

Insect Cage Experiments

The type of insect cage used in this study has a wire frame-work covered with sheeting muslin, into which are sewn two celluloid windows, 6 × 14 inches. The cages proved effective in preventing the movement of aphids in or out, and enough light is admitted to permit normal plant growth.

TABLE 3

Percentage of mosaic in plots 4, 4′ and 4″ in each row on five successive dates
(These plots are adjacent to plots having seed beets)

	Row	16	15	14	13	12	11	10	9	8	7	6	5	4	3	2	1
Plot 4	July 3	3.8	1.6	0.9	0.8	2.5	1.5	0.0	3.4	3.7	0.8	1.6	1.9	0.9	1.5	1.8	2.6
	July 14	3.8	1.6	3.6	1.6	4.1	3.8	3.2	3.4	5.1	4.8	3.3	1.9	4.9	1.5	7.1	15.5
	July 29	10.7	5.6	6.3	7.2	7.4	6.0	5.6	8.4	16.3	6.2	13.2	13.0	12.1	18.8	20.3	36.2
	Aug. 21	18.3	15.2	18.9	15.2	16.0	17.2	15.3	21.8	28.1	22.4	29.6	30.4	37.8	45.0	50.4	73.3
	Harvest	52.6	30.8	46.3	39.8	46.3	43.5	43.5	48.7	67.4	59.4	63.6	64.8	71.7	84.2	80.5	92.2
Plot 4′	July 3	0.8	0.0	0.0	0.8	0.0	0.78	0.98	0.0	0.0	0.9	2.4	1.5	0.9	2.4	0.8	0.8
	July 14	2.4	0.9	0.0	1.6	0.0	1.56	0.98	0.0	0.0	3.6	2.4	2.3	5.4	8.8	2.4	8.8
	July 29	4.0	1.6	2.5	5.6	7.2	3.9	3.9	4.5	5.4	6.3	9.6	5.5	12.6	16.4	8.8	19.2
	Aug. 21	12.0	9.0	9.1	14.4	10.0	12.5	14.7	12.7	16.2	27.0	20.8	19.5	31.5	31.8	42.4	45.6
	Harvest	21.4	23.8	15.3	25.4	21.8	35.3	26.2	34.5	33.6	46.2	37.6	41.4	40.0	57.4	60.6	79.8
Plot 4″	July 3	0.0	0.8	0.0	0.9	1.7	0.8	0.0	0.9	0.9	1.6	0.8	2.5	0.0	0.0	2.4	2.9
	July 14	0.0	2.4	1.0	1.8	2.6	1.5	1.7	3.8	2.8	3.3	2.3	3.3	1.9	2.9	2.2	10.3
	July 29	4.8	13.2	6.0	5.8	7.0	3.3	13.9	6.7	14.1	8.1	8.4	12.4	15.0	17.6	26.6	34.0
	Aug. 21	15.2	19.0	11.0	16.5	14.8	13.4	22.6	21.9	27.3	18.7	20.6	26.4	26.4	31.6	43.5	53.3
	Harvest	26.4	38.0	29.0	35.9	30.4	32.8	46.0	40.9	44.3	37.4	39.7	50.4	46.2	63.9	63.7	79.2

The common greenhouse aphid (*Myzus persicae*) was used. The initial brood was secured from carnations in a commercial greenhouse. The aphids were allowed to multiply on carnations in an insect cage.

Previously, roots were set in the greenhouse bed and caged. These included:

1. Beets which showed distinct mosaic symptoms at time of harvest.
2. Beets from commercial field and free from all symptoms.
3. Beets which showed no mosaic symptoms at time of harvest, but which grew in plots containing diseased individuals.

Beets in groups 1 and 3 soon developed mosaic symptoms in the greenhouses whereas those in group 2 remained normal. Aphids from the caged carnations were introduced on to one plant of each group, and after being allowed to feed and multiply for a period, were caged with healthy seed beets and healthy seedlings. Limited space in the greenhouse necessitated the use of a small number of cages.

Aphids which fed on the seed beet (group 1) with mosaic were transferred to three healthy seed beets on December 14. On January 6, all had distinct mottling.

Aphids which fed on the seed beet free from mottling (group 2) wer

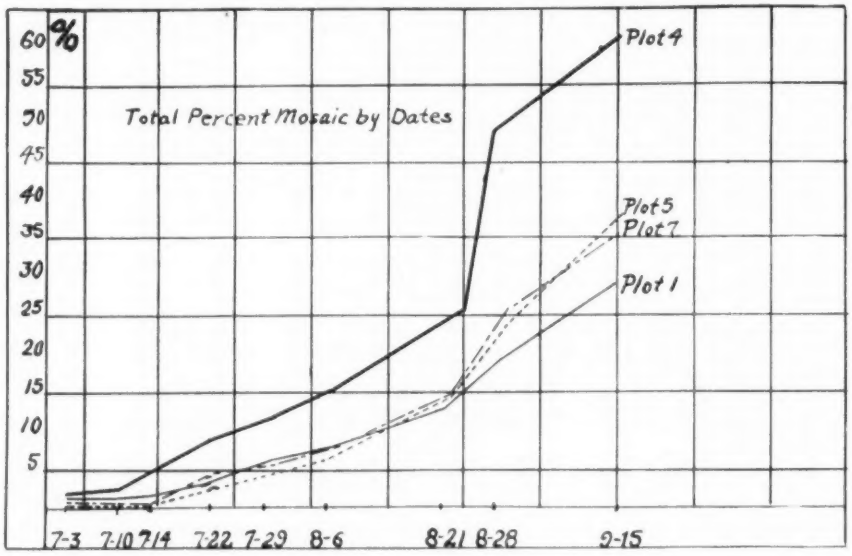

FIG. 8. PROGRESS OF MOSAIC IN FOUR ROTATION PLOTS AS THE SEASON ADVANCED

transferred to one healthy seed beet on December 14. The aphids fed and multiplied rapidly. No mosaic symptoms developed.

Aphids which fed on the mosaic seed beet of group 3 were transferred to a healthy seed beet on December 14. On January 6, unmistakable mosaic symptoms were in evidence.

Aphids which fed on the seed beet (group 1) with mosaic were transferred to two caged pots containing 7 seedling plants on January 4. On January 22, 6 plants had developed mosaic.

Aphids which fed on the seed beet (group 2) free from mottling were transferred to two caged pots containing 10 plants on January 4. All plants remained normal.

Aphids which fed on the seed beet (group 3) with mosaic, were transferred to two caged plots containing 11 plants on January 4. On January 22, 8 plants had developed mosaic.

Experiments on insect transmission were repeated; aphids from diseased and healthy plants were transferred to seedling beets March 7. The check plants remained normal. Those to which aphids from diseased beets were transferred had distinct mottling on March 21. This gives an incubation period of 14 days which is considerably shorter than that obtained in the previous test. This is probably due to the temperature in the greenhouse which was somewhat higher for the latter period. A third series of insect inoculations was started on March 17. Mottling was in evidence March 29. This gives an incubation period of 12 days.

At the present, other insects which frequent the sugar beet are being tested as possible conveyors of mosaic.

Artificial Inoculations

Up to date, all attempts to transfer the disease by means of needle inoculations, by the injection of freshly prepared juice with a hypodermic needle, and by the insertion of freshly crushed fragments of mosaic leaves into slits on the leaf petioles and crown, have been unsuccessful.

Seed Transmission

Up to this time, there is no evidence of seed transmission of sugar beet mosaic. Lind (4) states that the mosaic disease of garden beets is not conveyed by the seed.

In 1920, seed was collected from a plant growing on a tract west of Denver, Colorado, the leaves of which were extremely heavily mottled, crinkled, thick and brittle. The mottling extended upward to include not only the floral bracts, but the perianth segments as well.

This seed was threshed by hand, and a hundred or more of the seed were planted in pots in the greenhouse. The pots were caged. All seedlings were normal, showing no evidence of mosaic. It was anticipated that although there might be no embryo-transmission, the disease would be conveyed by the adhering perianth but such does not seem to be the case.

WINTERING-OVER OF MOSAIC

A steckling may carry the infectious principle or virus. Roots from plants which had mosaic were topped and placed in a cool cellar to undergo a period of rest. When such roots were set out in the greenhouse the very first leaves to unfold were characteristically mottled and often malformed.

As has been pointed out, plants apparently disease-free at harvest time and which had been so throughout the season may be carriers, as is evidenced by the early development of symptoms on the roots when set out.

A high percentage of the seed beets on the experimental farm of the Great Western Sugar Company in 1920, which came from diseased steckling fields of 1919, were clearly and unmistakably diseased, as was indicated by the mottling and puckering of the first leaves within a few days after these unfolded. In fact, mottling could often be detected on a leaf not yet fully unrolled by spreading it open.

If the disease is seed transmitted, this is very rarely the case.

There remains the possibility of wintering-over on such weeds as *Chenopodium album* and Amaranthus spp., and in the soil, neither of which possibilities have been investigated thus far.

CONTROL

It has been demonstrated experimentally that the infectious material may be carried by aphids from a diseased to a healthy plant. There is no evidence as yet that there is embryonic transmission of the infective principle. Up to this time the disease is found chiefly in breeding plots, seed fields and steckling fields. Clearly, stecklings are inoculated by insects flying from near-by diseased seed beets. The virus retains its vitality in the steckling throughout the silo period, and the resulting seed plant develops the disease. These affected seed plants are in turn centers of infection. Of course, a diseased commercial beet also may be a center of infection.

To have disease-free seed plants we must have disease-free stecklings. This means that steckling fields should be such a distance from seed fields that there will be little chance of insects conveying the inoculum. The past season we have observed transmissible mosaic on commercial beets and stecklings to a maximum distance of 1½ miles from diseased seed beets. However, the diseased plants at that distance were very few in number.

SUMMARY

1. Mosaic of sugar beet has become increasingly prevalent the last few years in steckling and seed beet fields of northern Colorado and

western Nebraska. It also occurs in commercial beet fields near a possible source of infection.

2. Mosaic of sugar beet is distinct from curly-top of sugar beet.

3. The principal symptom is mottling of the leaves, which may or may not be associated with their malformation.

4. Aphids carry the infectious principle.

5. Under greenhouse conditions, an incubation period on seed beets of approximately 24 days has been established; on seedling plants from 12 to 18 days.

6. Thus far, evidence of seed transmission is lacking.

7. The virus retains its vitality in the steckling throughout the silo period. This is the only means of wintering-over thus far known.

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BITTER PIT IN APPLES AND PEARS: THE LATEST RESULTS IN PREVENTIVE MEASURES

D. McAlpine

After five years' continuous experimental investigation into the cause of bitter pit, and having regard to any remedial measures suggested thereby, I believe that it would facilitate further research to state as briefly as possible what has been done and what remains to be done. Five voluminous reports have been issued,[1] and since these reports are available at the Commonwealth Department of Trade and Customs for anyone defraying the postage thereof, it is necessary for me to state only the essential facts and the details can be filled in by anyone who is sufficiently interested to refer to the reports themselves.

In dealing with this investigation from the Australian point of view, there were two chief problems to be solved. First and most urgent considering the loss sustained, it was necessary if possible to prevent the development of the trouble during shipment overseas. Second, to prevent the development of the disease in the orchard while the fruit was still growing.

BITTER PIT IN RELATION TO COLD STORAGE

After several years' experiment, the first problem was satisfactorily solved, and apples and pears can be carried to the other side of the world and reach their destination in the same condition as when put on board ship. It has now been determined that this immunity from disease not only applies to bitter pit, but from the nature of the process, the fruit is kept in a state of suspended animation so that no change occurs and no other injurious disease develops. It has also been settled by actual experiment how long the fruit retains its immunity after being removed from cold storage, so that there is sufficient time to market the fruit before deterioration occurs. All the details are given and the precautions to be taken as regards the picking of the fruit, etc., in the published reports.

The essential part of the process is stated by Keeble[2] as follows: "As the result of experiment, he shows that if apples be stored at a temperature of about 30° or 32° F. and if fluctuations beyond these limits be prevented, no bitter pit manifests itself during a period sufficiently pro-

[1] McAlpine, D. Bitter pit investigations. Progress Report **1**: 1–197 1911/12; **2**: 1–224. 1912/13; **3**: 1–176. 1913/14; **4**: 1–178. 1914/15; **5**: 1–144. 1915/16.

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longed to transport the fruit from Australia to Europe. This is a great gain" . . . It is very encouraging to find that this method is at last being adopted in Australia in the numerous cool stores dealing with apples and pears. The oversea shipments of these fruits are also being carried out on the principle laid down and the best evidence of practical success lies in the condition in which such consignments are landed. I need hardly point out that the same principle may be applied to other fruits which have hitherto failed to carry when the freezing point of such fruits has been determined.

CAUSE OF BITTER PIT

Before dealing with preventive measures in the orchard, it is desirable to state briefly what we know as to the cause of the disease. It would have simplified matters immensely if any definite organism had been discovered, but careful and continuous investigation has shown that insects, fungi and bacteria must be excluded. The theory that spraying with poisonous materials, such as arsenate of lead, is responsible for bitter pit has been definitely disproved. In seeking for the cause, we are therefore thrown back upon the structure and working of the tree, and more particularly of the fruit, where the disease manifests itself.

From the point of view of bitter pit, the most important structure in the apple or pear is the continuous vascular network immediately beneath the skin, where bitter pit originates. This network is discernible even in the very youngest stages of the fruit. It forms the boundary between the pulp and the skin, and has the important function of regulating the supply and distribution of the sap, to nourish the increasing pulp cells and the expanding skin in the growing apple or pear. We can now understand why this regulatory system of vessels exists, and how if it is interfered with in any way, a derangement of the tissues takes place. Wherever bitter pit occurs this vascular network is ruptured and the pulp cells there have become brown and dry, resulting in the consequent falling in or depressions in the skin which constitute the external symptoms of the disease.

We can now picture to ourselves how bitter pit originates. When there is an extra rush of sap, following on dry conditions, the rapidly swelling pulp cells at the external boundary burst the vascular network at localized spots, and the sap pressure which is sufficient to rupture the enveloping network, also bursts the thin-walled pulp cells at these particular spots and death of the cells ensues. Briefly, it may be stated that rapid alternations between dry and moist conditions, combined with fluctuating temperatures, during the growing stages of the fruit, is the exciting cause of bitter pit. Hence, the inherent difficulty of the task

of preventing bitter pit in the orchard in a locality subject to such fluctuating conditions of humidity and temperature.

PREVENTIVE MEASURES

It will be readily understood from a consideration of the causes which produce bitter pit that there is no royal road to its prevention in the orchard. Light pruning in contrast with severe pruning was shown to reduce the amount of disease considerably; and in irrigated areas a light watering throughout the season, instead of a heavy watering towards the end of the growing period has beneficial results. It was recognized at an early period of the research that there were three main lines of investigation worthy of being followed as a probable means of minimizing the pit:

(a) Experiments with different stocks.

(b) Crossing liable and non-liable varieties.

(c) Breeding of bitter pit-proof varieties by selection and crossing.

(a) *Experiments with different stocks.* The question of the best stocks to use for fruit trees is constantly cropping up and the Australian orchardist is very desirous of scientific guidance in the matter. At the recent meeting of the Australian Association for the Advancement of Science in Melbourne a paper was read on "Apple-tree Stocks," and as showing that this important branch of fruit-growing has not hitherto been made the subject of exact experiment it was strongly urged that the matter should be made the subject of investigation. The "blight-proof stock" is used for apples and it seemed desirable to test the possibility of a "pit-proof stock," that is to say, stock and scion so intimately knit together that the "flow of sap" is regulated and controlled. There is one direction in which the stock undoubtedly affects the scion and that is in the supply of water. This fact may have an important bearing on the pit problem, and it was clearly shown in an experiment where varieties of apple on Winter Majetin stocks required artificial watering while the same varieties growing alongside on Northern Spy stocks were luxuriant and thriving, without any artificial aid. Of course the varieties were all of the same age. In the first year of the investigation, 56 apple trees, mostly susceptible varieties, were budded or grafted on a number of different stocks and planted in a bird-proof enclosure. These were practically all treated alike and the primary object was to test the effect of the stock on the tree, as regards bitter pit in the fruit. Other points of interest were observed at the same time, such as the effect of the stock on time of flowering, on fruiting, on the vigour of the tree, etc., but the main points to be determined were, which stocks were associated with the least amount of pit, the largest yield, and the most vigorous tree. In

the last year of the experiment when the trees were five years old, i.e., from the date of planting, there were great variations in the yield as might be expected. Cox's Orange Pippin topped the list with a yield of 173 apples, and it was interesting to observe that the same variety growing alongside only yielded 7 apples. In the case of the small yield, the stock was Winter Majetin and none of the apples were pitted; while in the case of the large yield, the intermediate stock was French Paradise on Spy roots and there was 44 per cent of pit. The small yield of fruit and the poor growth of the tree indicated that the Winter Majetin stock was evidently unsuitable under the given conditions. When this stage was reached, the experiments were abruptly stopped, in spite of the representation of practical fruit growers who contended that at least ten years wou'd be necessary from the date of planting to test the influence of the stock on the amount of bitter pit. Fortunately, however, a valuable series of stock experiments were being carried out by Mr. Quinn, at the Government Experimental Orchard, South Australia, and these are still being continued through the agency of that State.

The results for the past season are just to hand, and since the trees are now in full bearing, the evidence is beginning to accumulate as to the value of this means for minimizing the prevalence of bitter pit. The report states: "There is some slight evidence to indicate that the use of an intermediate stock between the Spy, the Winter Majetin, or Cole's blight-proof roots, tend to diminish the prevalence of bitter pit in the fruit. But a great number of crops will require to be harvested before any pronouncement can be made in this respect."

At the Burnley Horticultural Gardens, Victoria, there were various other stock experiments initiated. For instance, Cleopatra (syn. Ortley), which is very liable with us, was budded and grafted on to the pear stock. This stock was chosen because the pear was generally free from the disease in this particular locality. Crab stocks were also used. In order to make certain that the seedlings used were from wild trees, I had pips sent out from Britain along with stocks of the wild crabapple. The seeds were planted in a cold frame and produced vigorous seedlings.

(b) *Crossing liable and non-liable varieties.* A beginning was made by taking a susceptible variety such as Cleopatra and crossing it with a comparatively non-susceptible variety such as Yates. It may be noted that both these varieties are of American origin and have been imported here. The seeds of the resulting cross were sown in a pot, and the one plant obtained was planted out.

(c) *Breeding of pit-proof varieties by selection and crossing.* The breeding of perennial plants, such as fruit trees, is a big undertaking and involves a considerable amount of time, in order to reach results of com-

mercial value. But just as breeding stations for annual plants, such as wheat, have been established, so it is becoming more apparent that fruit trees must receive similar attention, not only for the production of new and better varieties than those at present cultivated, but for the scientific treatment of various serious diseases to which they are subject. And there is no disease for which it is more necessary to alter the constitution of the tree than that of pit. It is appropriately called a constitutional disease, since the root of the trouble really lies in the artificial nature of our modern apple. When one considers that the plump succulent and sweet cultivated apple has been derived from the small and sour crab, and that in this process the hardy nature of the ancestor has been sacrificed, particularly in the direction of a softening of the fibre, it must be confessed that the penalty paid for the increased attractiveness is a weakening of the constitution. It is only by systematic breeding that a type of apple can be secured with suitable arrangements for the preservation of moisture in dry air and for resisting sap pressure in moist air; and I am fortified in this conclusion by the recommendation of Luther Burbank: "From your report (on bitter pit) I am more than ever convinced of what I have long believed, that all fruit diseases and defects must in the end be *bred out* of them, rather than combated in varieties which are susceptible *to* them."

I have given this brief summary of a very extensive investigation in the hope that it may induce those who are already engaged in the scientific investigation of fruit tree stocks, or in the breeding of new varieties of fruit trees, to give attention to this particular disease in apples and pears, while carrying out their general plan. It is only by the cooperation of various workers that such a constitutional disease can be thoroughly understood, and the means of preventing or minimizing it discovered.

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A NOTE RELATIVE TO THE RECENT APPEARANCE OF THE SUGAR CANE DOWNY MILDEW IN THE PHILIPPINES

WILLIAM H. WESTON, JR.

The disease of sugar cane caused by the conidial Sclerospora, *S. sacchari* Miyake, and known by such names as leaf-stripe disease, Queensland (or Australian) leaf-stripe, cane-dew fungus, and downy mildew of cane, has had an interesting history. Although this disease occurred and was recognized as a disease in Queensland, Australia, in the Fiji Islands, and in Formosa as early as 1909, no published reference to it was made until June 1911. At that time Lyon (3) briefly mentioning the disease as a "leaf-stripe disease" of sugar cane, described the characteristic striping of the leaves and sheaths, and the conspicuous abnormal elongation of the stalks which he illustrated with a photograph, and characterized the disease, of which the cause had not then been accurately determined, as a "quite infectious and quickly fatal" one, that might at any time become a "very serious factor in the sugar industry of Fiji." In March 1912, Miyake (5), who had been studying the disease since 1909 when it appeared at the Formosan Sugar Experiment Station in plots grown from cuttings imported from Queensland, published a detailed report of his work and established the causal fungus as a new species, *Sclerospora sacchari*. His paper was a very comprehensive one, discussing at length the symptoms of the disease, the nature, relationships, and effect of the causal fungus, his experiments on its means of infection and spread, and the progress of attempts to control it in Formosa. Unfortunately, the bulletin was in Japanese and so was unavailable to most pathologists. In November 1913, however, Ito (2) briefly mentioned Miyake's paper, giving a Latin diagnosis of the causal fungus, and urging that a new sub-genus be established to include this and other Sclerosporas in which the conidia when germinating, form tubes instead of the zoospores which characterize the generic type. In April 1915, Lyon (4) gave in English a valuable account of the disease based on a translation of Miyake's article, supplemented by his own experience with the disease in Fiji and Queensland, and by information and material received from Mr. D. S. North of the Colonial Sugar Refining Company who had observed the activity of the disease in both these regions. Alarmed by an increasing knowledge of the menace of this disease, the Federal Horticultural Board of the United States Department of Agriculture decided to take further precautions to protect the

United States in addition to the earlier Quarantine Orders 15 and 16 which prevented the importation of sugar cane from any foreign country and from Hawaii and Portò Rico. Accordingly, as Miyake in Formosa had found that the disease was as destructive to maize as to cane, and hence might be introduced on that host also, the Federal Horticultural Board in July 1916 established Quarantine No. 24 which broadened the prohibition of an earlier order (Quarantine No. 21 of March 8, 1915, preventing the importation of maize from parts of the Orient harboring dangerous maize mildews) and prevented the introduction of maize or related plants from oriental countries where the disease was known or suspected to exist.

In view of the fact that this disease was known from such important cane-growing centers of the Orient as Formosa, Queensland, and Fiji, the writer, while in the Philippine Islands from May 1918, to March 1920, made a careful search for it there but in vain. In the Island of Cebu the writer encountered one sugar-cane clump which had been infected by a conidial Sclerospora (*S. spontanea*) from a badly infected cornfield nearby (6). Also while searching in the mountainous interior of Luzon for the oogonial stage which he believed might occur on sugar cane or its wild relatives at that higher altitude, the writer in January 1920, found an oogonial Sclerospora which was severely injuring the native-grown sugar cane at Bontok, and made the first collections of this fungus, which hitherto had been unknown in the Philippines (6). No instances of infection by *Sclerospora sacchari* were noted, however, although this disease was searched for with care over large areas of cane-growing country from the Visayan Islands in the south to the northern part of Luzon.

Obviously it was a matter of vital importance to the valuable sugar industry of the Philippines that the islands should continue to keep out this dangerous disease from which at the time they were so fortunately free. While returning to the United States in April 1920, however, the writer learned from Mr. C. W. Hines, formerly of the Philippine Islands Bureau of Agriculture, that a Japanese company owning a large plantation near Manila, had imported a short time before over 70 sacks of sugar-cane cuttings from Formosa to use for planting; and that the Philippine Bureau of Agriculture not only had permitted this importation but had secured from the Japanese company a number of cuttings of several desirable varieties for propagation and subsequent distribution. Alarmed at the strong probability that the Formosan downy mildew might thus be introduced into the Philippines, the writer in November 1920, sent this information to Mr. H. Atherton Lee, who had recently been appointed as pathologist with the Philippine Bureau of Agriculture, and

urged that plantings from these cuttings be watched for the possible development of downy mildew. In January, 1921, Mr. Lee replied that no Sclerospora had appeared as yet, but letters recently received (written April 28 and May 12) bring the disquieting information that a conidial Sclerospora has appeared on Formosan cane on the Japanese plantation at Payatas, Rizal Province. Although Mr. Lee has made only a preliminary study of the causal fungus itself, and has devoted his efforts primarily to eradicating the disease, the evidence is strong that the downy mildew is none other than *Sclerospora sacchari* Miyake, which has thus been introduced from Formosa and started in the Philippines. In his letter of May 12, Mr. Lee says, "The conidia of the Payatas cane mildew are not elongate, as in the case of *S. spontanea*, they are more oval with ends more sharpened than smoothly rounded. Moreover, . . . our cane mildew has been found only on the imported Formosan canes. Native cane fields on three sides of the Formosan cane have so far been absolutely free from infection."

The situation has two important aspects. In the first place, the fact that the disease occurs in the Philippine Islands is of considerable interest to other sugar-growing countries. To them, the history of the introduction of this disease into the Philippines teaches a valuable lesson in that it shows how easily the disease may enter a country in spite of inspections and other precautionary measures. As Mr. Lee remarks in a manuscript note[1] enclosed in his letter of April 28, "The sugar cane points, according to the Japanese firm, had been grown by the experiment station of the Japanese Government in Formosa," where they had been dealing with the disease since 1910, and would be best qualified to select healthy cuttings. Not only this but, as an added precaution, the cane was allowed to enter the Philippines only "after dipping in Bordeaux mixture." Furthermore, it was early in 1920 that the cane was imported and planted, but although "upon the appointment of the writers (Mr. Lee and Mr. Medalla) to the plant disease laboratories in March 1920, they became cognizant of these circumstances, and since then, periodical inspections of the plantings have been made," it was not until "April 1921, the cane having been ratooned (that) numerous cases of etiolation of the young plants were observed." The circumstances of this introduction, as well as those attending the introduction into Formosa [cf. Miyake (5) and Lyon (4)], show that the disease is an especially dangerous one; because it is carried in cuttings, and is so insidiously persistent in cane as to make selection of healthy cuttings extremely difficult even where the disease is recognized; because it is almost impossible to detect in cuttings on inspection, and is unaffected by their treatment with Bordeaux; and

[1] Published in Science, N. S., **54**: 274–275. Sept. 23, 1921.

because in developing cane the disease for a long time remains latent or at least sufficiently obscure as to escape notice. A full realization of the manner in which this disease has invaded the Philippines may afford other cane-growing countries a basis for quarantine restrictions and other methods of protection. As far as the continental United States is concerned, little anxiety need be occasioned by this new development in the cane disease situation, because existing quarantines provide ample protection, and because the procedure by which desirable new canes are introduced by the Department of Agriculture provides that cuttings be grown in quarantine greenhouses under surveillance long enough to be quite sure the resulting plants are healthy before they are released. Provision should be made, however, so that no chances may be taken of introducing the disease from the Philippines into Guam, the Virgin Islands, Porto Rico, Haiti, the Dominican Republic, and the Canal Zone, or any other cane-growing regions which are not adequately protected by existing measures but with which the United States is concerned in a controlling or advisory capacity.

The second important aspect of the situation is that the establishing of the disease in the Philippines constitutes a grave menace to the sugar industry of the islands. In the agriculture of the Philippines, sugar cane is a major crop, and the sugar industry is very important financially, the value of the investments involved in the crop (which was worth $32,231,404 in 1919) being considerable. The previous history of this downy mildew in Formosa indicates that in the Philippines also it probably will prove to be a serious factor. In Formosa, by a most stringent eradication campaign enforced by the Government, the downy mildew was reduced to a minimum on sugar cane; but it spread to maize and proved to be even more destructive to this host, with the result that attempts to control it were opposed by practically unsurmountable obstacles. It seems highly probable that in the Philippines the problem of controlling the downy mildew will be a serious one. Not only is maize exceedingly common throughout the islands, but teosinte (which Miyake inoculated with equal ease) is grown occasionally; while everywhere throughout the length and breadth of the archipelago are found at least two species of wild Saccharum and several other grasses so closely related to sugar cane that it seems inevitable some of them will prove susceptible to the disease. On the other hand, it is possible Philippine conditions may be such that spread of the disease from sugar cane to maize may not readily take place there. Professor W. L. Waterhouse of the University of Sydney has informed the writer that, although he has seen the downy mildew on sugar cane in both Queensland and Fiji, he has never seen maize attacked by it in either of these localities despite ample opportunity for such inocula-

tion. Moreover, although the disease is probably still present in those regions, it has not caused sufficient devastation to occasion published notice, nor has it, if Haywood's account (1) may be relied upon as conclusive, spread so widely as to extend from Queensland to the sugar cane growing coastal strip of northern New South Wales.

In the Philippines the difficulty of control, besides being aggravated by the presence of alternate hosts, will also be increased by the fact that cane for the most part is not concentrated in large easily-controlled holdings, but is grown in small areas by many independent planters, large numbers of whom contribute to a single "central" mill. For this reason and because of the nature of the government in the islands, absolute government control of the disease situation such as was practiced in Formosa, will be impracticable in the Philippines.

Fortunately, however, the potential menace of this outbreak of sugar-cane mildew in the Philippines is well realized there, and since Mr. Lee has begun an immediate and drastic campaign of eradication while the infection is still confined to a few localities, there is every hope that by vigorous efforts the rapid control or even complete eradication of the disease may be accomplished.

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PHYTOPATHOLOGICAL NOTES

Plant disease survey of England and Wales.—That there is in existence a plant disease survey of England and Wales which publishes an annual report may not be generally known to pathologists of North America. The publication is issued by the Ministry of Agriculture and Fisheries located at 10, Whitehall Place, London, S. W. I. The title of the latest report is "Report on the Occurrence of Insect and Fungus Pests on Plants in England and Wales for the Year 1919." It is a modest pamphlet of 68 pages. It is known as Miscellaneous Publication No. 33. Similar reports for 1917 and 1918 have been issued. They are Miscellaneous Publications 21 and 23.—PERLEY SPAULDING.

Summer meeting of the American Phytopathological Society.—The annual summer meeting of the American Phytopathological Society was held at St. Paul, Minnesota, and Fargo, North Dakota, July 19 to 22, inclusive, in conjunction with the conference of cereal pathologists. There were present as guests of the Society, Dr. E. J. Butler, Imperial Bureau of Mycology, London; Dr. Kingo Miyabe, professor of botany and director of the Botanic Garden, Hokkaido Imperial University, Sapporo, Japan; Mr. R. J. Noble and Mr. James P. Sheldon, Department of Agriculture, New South Wales, Australia.

Prof. A. Jaczewski, director of the institute of mycology and phytopathology, Petrograd, Russia, and Prof. N. I. Vavilov, Bureau of Applied Botany and Plant Breeding, Petrograd, Russia, arrived in America too late to attend the conference. They made an extended tour of the United States and Canada before returning to Russia.

There were present members of the Society from the Philippine Islands, Saskatchewan, Manitoba, and Ontario, Canada, and from the following states: Minnesota, Wisconsin, North Dakota, South Dakota, Kansas, Nebraska, Indiana, New York, Delaware, and District of Columbia.

The days were fully occupied with visits to the experimental plots on the station grounds and the evenings with round table discussions. The following subjects received special consideration at the conferences: Root and ear rots of maize; downy mildew of maize and cereals; root rots and "take-all" of cereals; flag smut of wheat; barberry eradication; wheat rust investigations.

Owing to the very dry and hot weather of the preceding weeks the experimental plots were farther advanced than usual and some of them were ready for harvest. Stem rust was not abundant but was present in

sufficient amounts to show the difference between the susceptible and resistant varieties of wheat. Stem and root diseases of cereals were found plentifully in both localities.

The local committees at St. Paul and at Fargo made excellent provision for the comfort of the visitors and afforded every facility for a profitable and enjoyable meeting. Appropriate resolutions were adopted to express the appreciation of the visiting members.

In view of the report on the status of barberry eradication a resolution was adopted endorsing a vigorous campaign to hasten the work.

The first Cereal Disease Conference was held at St. Paul six years ago At this first meeting Dr. F. Kölpin Ravn was an honored guest. In view of this a letter was drafted and ordered sent to Mrs. Ravn to express sympathy at the untimely death of her distinguished husband.

Bibliographie der Pflanzenschutzliteratur. Das Jahr 1920.—This is a new indexing periodical issued by the Biologische Reichsanstalt für Land. und Forstwirtschaft in Berlin-Dahlem. The number for 1920 is the first one issued but there is to be another for the years 1914 to 1919. Hollrung's Jahresberichten über das Gebiet der Pflanzenkrankheiten was discontinued with the number for the year 1913 and the present less pretentious publication takes its place. A casual inspection and comparison of the two publications does not show to the writer any essential difference between the fields covered by the two. The Bibliographie is purely an indexing organ and is in slightly more compact form than Hollrung's Jahresbericht. The former contains 71 pages and approximately 1800 references as compared with 2360 references in the last number of the latter. The arrangement of the material is somewhat changed but is even more usable than the old. The publication is intended as an exchange organ; its price when sold is unknown to the writer. The same institution is beginning the publication of an 8-page folio monthly entitled "Nachrichtenblatt für den deutschen Pflanzenschutzdienst."—PERLEY SPAULDING.

Citrus canker in the Hawaiian Islands.—Citrus canker has been found in two orchards in the Hawaiian Islands. One of these orchards is located in Honolulu, Island of Oahu, and the other at Kilauea, Island of Kauai. Citrus growing is of very minor importance in the Hawaiian Islands and such species and varieties as are commonly grown are not extremely susceptible. For these reasons it has not been considered advisable to recommend the application of the Florida methods of eradication of this disease. It seems desirable, however, to call attention at this time to the presence of the disease in Hawaii in order that the fullest protection for the citrus industry of California and the southwestern states may be obtained.—H. L. LYON and H. ATHERTON LEE.

Sclerotium rolfsii on velvet beans.—Specimens of velvet beans attacked by *S. rolfsii* were recently received from Altamaha, in southeastern Georgia, showing the characteristic symptoms of this rather general disease on a hitherto unreported host. The trouble was reported as new to the neighborhood where it was found. The plants killed by the disease occurred promiscuously in the field and not continuously in the rows. The leaves of the affected plants were dead, dry, and brown, and the stems almost coal-black. On the main tap root the weft of fungus hyphae extended 2–4 inches below the surface of the soil and about ½ to 1 inch above. On most plants considerable mycelium was present on the outside of the stem and roots, and in some cases sand was held around the stem by the mycelium, forming a very noticeable enlargement. Several to many round, white to brown sclerotia about the size of mustard seed were found on some plants. The sclerotia were produced at numerous points but more abundantly just above the soil surface (Fig. 1).—W. W. GILBERT.

FIG. 1. Velvet bean plant killed by Sclerotium rolfsii: Note abundant weft of mycelium white above, brown below with numerous white immature sclerotia in area indicated by X.

Disinfecting laboratory cutting instruments.—In an article entitled "Field Cultures of Wood-Rot Fungi on Agars"[1] Bessie E. Etter recommends heating the scalpel or scissors red hot before each inoculation. The writer has employed this method but it was unsatisfactory because the scalpels were soon made worthless from heating. In making isolations of organisms from fruits he has employed the following method very successfully:

After scraping off any debris which may be adhering to the surface of the cutting tool, the tool is plunged into a wide-mouthed bottle of 90 per cent ethyl alcohol in which it is left a minute or more and then the alcohol burned off but care being exercised not to heat the tool enough to injure it. Christiansen[2] found that one-half a minute was sufficient time for disinfection to be accomplished with 70 to 90 per cent ethyl alcohol, the lower and higher percentages of alcohol requiring a somewhat longer time.—J. S. COOLEY.

[1] Phytopathology, **11**: 151–154. 1921.

[2] Johanne Christiansen. Zue Theorie und Praxis der Alkoholdisinfektion. Zeitscrift für physiologische Chemie. Hoppe-seyler **102**: 275–305. 1918.

Sunburn and tomato fruit rots[1].—The sunburn of tomatoes is very common on fruits which are more or less exposed to the sun during very hot weather, and is especially severe on fruits where the vines have been defoliated by *Septoria lycopersici* or other causes which results in their exposure to the sun. Sunburn is most severe on fruits which are approaching maturity and just beginning to color. The sunburnt area does not assume the normal red color of a ripe fruit but takes on a slightly reddish yellow. The varying amount of red and yellow in the sunburnt areas depends on the ripeness of the fruits at the time of injury.

These sunburnt areas develop numerous very small, light brown spots which are referred to by the growers as "freckles." In the beginning, these spots are mere pin points but they increase in size, become black and finally rupture, releasing great numbers of spores of Alternaria. These spores are extremely variable in size but a very large number of them show characters which justify classification as *A. solani* (E. & M.) J. & G. In some cases these spots rupture when about ¼ inch in diameter while in other cases they coalesce and cover practically the entire sunburnt area. (See figures 1, 2, and 3).

The question very naturally arises as to whether these spots are due to the pathologic condition resulting from the sunburn or to the fungus which is found growing in them. A large number of sunburnt tomatoes were gathered, wrapped separately in paper and brought into the laboratory. These were grouped with reference to the size of the spots into groups a, b, c, d, group "a" having the smallest and group "d" the largest spots.

The epidermis was peeled back and cultures prepared by touching a sterilized needle to the pulp below the spot and then to the agar in a petri dish. These cultures gave no growth from group "a," good growth from most of group "b" and from all of groups "c" and "d" indicating that the spots were formed in advance of the infections.

Pure cultures of the organism were applied to the uninjured surface of sunburnt areas and to the surface of normal ripe fruits; also to the surface of normal ripe fruit in which the epidermis had been punctured with fine sterilized needles. The checks consisted of the same kinds of fruits but they were not inoculated. In all cases the surfaces of the fruits were sterilized before puncturing and before inoculation. Of course, it was impossible to determine whether these fruits had been infected before gathering or not. The results of these tests indicated that the organism attacked fruits that had been injured by sun burn or by puncturing with a needle very readily but that it attacked the normally ripened uninjured fruits very slowly.

[1] Paper 62 of the Journal Series, New Jersey Agricultural Experiment Stations, Department of Plant Pathology.

This was followed by a study of the epidermis of the spots as compared with the epidermis outside the spots. These studies showed, 1. that the spots were due to a discoloration of the epidermal cells and that these spots could increase in size in the sunburnt areas indefinitely regardless of the presence or absence of the organism and, 2. that this was followed by a pulling apart or slight rupturing of these darkened cells. This very naturally produced most favorable conditions for natural inoculation with the spores of the fungus.

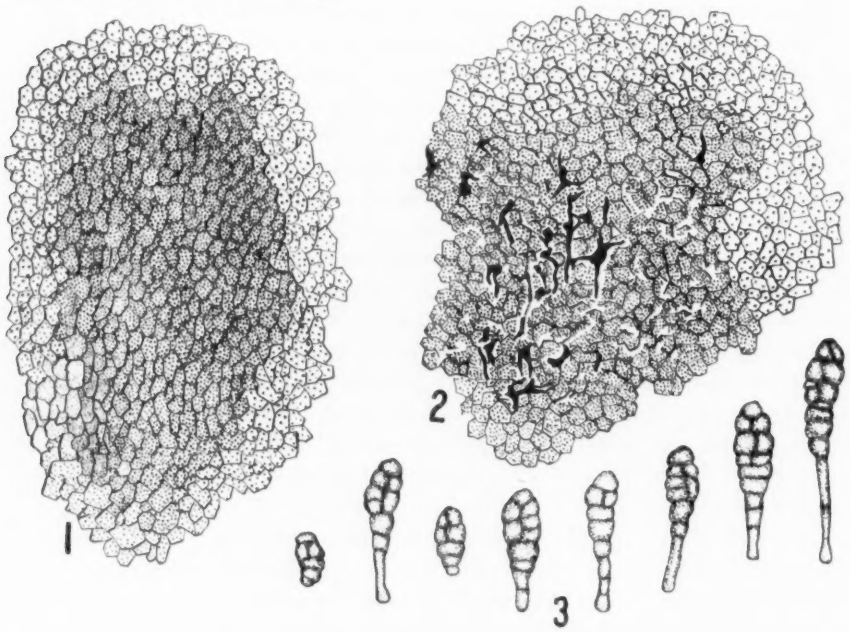

FIG. 1. Brownish spot on sunburnt fruit before rupturing of the epidermal cells.
FIG. 2. Spot on sunburnt fruit showing the rupturing of the fruit.
FIG. 3. Spores from the infected spots. Drawings by Miss G. E. Macpherson and Mr. G. W. Fant

These studies indicate that sunburnt fruits are far more susceptible to fungus injuries than normal ripe fruits; and that the fungus gains entrance thru wounds, in this case ruptures in the epidermis which are the direct result of sunburning. This organism also infects the fruit cracks and causes rottings which result in great losses.—MEL T. COOK.

Leaf curling in tomatoes.—The accompanying illustration represents very clearly a phenomenon assumed by some tomato plants, about which inquiries have become very numerous of late years. (See figure 1).

Some years ago (in 1916) I observed this phenomenon in plants growing in Lethbridge, Alberta. Since then on numerous occasions my attention has been called to it. Indeed, European literature has dealt several times with apparently the same phenomenon.

The curling of the leaves is confined to plants grown as single stems and tied to stakes or other supports. It occurred in all varieties under observation,—Earliana, Bonny Best, Chalk's Jewel, Livingstone Globe,

FIG. 1. LEAF CURLING IN TOMATOES

John Beer, as well as in the yellow pear-shaped tomatoes, provided these plants are trimmed to one shoot. Last year records were kept of the yield of these plants. To begin with, the setting of the fruits was perfect, as near a complete set of fruits was observed as possible. The fruits were perfect in size and shape, and the yield very large. No injurious

influence was discovered. It was immaterial whether the plants trimmed to a single shoot were trained to stakes or left growing on the ground. Three rows of several varieties of tomatoes were planted out from seeds saved from plants showing pronounced curling. Every other plant was pruned throughout the season to one stem, the remainder were allowed to grow as they pleased. The effect was remarkable in as much as every pruned plant showed pronounced leaf curling, which was entirely absent in the unpruned plants. The comparative yields obtained were uniformly satisfactory in the pruned and unpruned plants. This year the same observation was made. The excessive and prolonged drought did not enhance the symptoms over the previous year, which was almost excessively wet.

The phenomenon is most pronounced; inquiries were received from many parts of Canada, and in all cases it was found that the plants were pruned to one stem. Correspondents stated that the yield was quite good. Nowadays one has become quite alarmed at noticing rolling of leaves in plants. This is no doubt due to the attention which leaf roll in potatoes has received. Indeed, the curl in tomato leaves resembles potato leaf roll very closely, especially in the firm succulent nature of the curled leaves. Anatomically the leaves of affected tomatoes resemble the structure with which we have become familiar in potatoes. The abundance of starch is remarkable in the curled leaves of both plants.

It was sought to determine whether mere increased "root pressure" could cause the curling. A carefully devised and checked experiment revealed that increased root pressure was well taken care of by the corresponding activity of the hydathodes of the leaves. No curling resulted, although the leaves were very firm, rigid and succulent. Eventually owing to lack of sufficient nourishment, the plants succumbed. It was thought that the removal of the side shoots diminished the area of the plant to such a degree that the ascending sap of a normal root supply might result in a mechanical curling of the leaves. The experiments conclusively showed this not to be the case. One observer (Poser, C. Gartenwelt **24**: 181. 1920) believes that the curling is due to excessive and sudden ventilation, which on correcting, prevented progress of the "disease." This must have been a wilting of greenhouse grown plants (as they were) when exposed to drafts of air.

Schoevers, T. A. C. (Tijdschr. over Plantenziekten **25**: 11-12, 1919) states that "pruning and stopping back" reduce development, hence the transport of reserve food materials from leaves to fruit is interfered with. This causes the non-parasitic rolling."

We have come to exactly the same conclusion. The phenomenon is not a disease, it does not interfere with the yield and quality of fruits

but it would indicate that tomato plants might well be pruned to two or three shoots, if it were proved that such method would give an increased yield.—H. T. GÜSSOW.

A "dry rot" disease of alfalfa roots caused by a Fusarium.—The pathogenicity of a root disease of alfalfa which is of considerable consequence to southern Utah and possibly to Arizona also, has been traced to the activity of a Fusarium. This fungus causes brown cankerous depressions to appear at the base of lateral roots, which it entirely destroys, and infests both the parenchymous tissue and xylem vessels of the tap root. The disease is most active during the hottest period of the summer and is apparently confined to hot dry localities. Plants attacked by the fungus wither and die with remarkable suddenness. The disease first manifests itself by a few dead or dying plants which soon become conspicuous in a green alfalfa field. These plants are centers from which the infection spreads in various directions, often destroying an entire field within a single season. Infected areas vary in size from a few feet to several rods in diameter, but always present a very striking and characteristic appearance. They are usually somewhat circular in outline and have at their outer margin an active zone about three feet wide which separates green, healthy appearing alfalfa from the black, decayed plants which have been killed by the disease. The outer margin of the zone is marked with plants just beginning to wilt. Back of these toward the devastated area are plants green but dry, indicating sudden death. The plants that form the inner rim of the zone are bleached to a straw color. Specimens of alfalfa roots recently received from Arizona gave positive evidence of the presence of a Fusarium similar to, if not identical with, the fungus which causes "dry rot" in southern Utah.—WALTER P. COTTAM.

Personals.—Dr. E. J. Butler, chief of the Imperial Mycological Bureau located at Kew, England, has been taking part in the Cereal Pathologists' Summer meeting. While in Washington, a smoker and lunch was held in his honor, thirty-two pathologists participating. In an informal talk Dr. Butler explained in some detail the aims and scope of work tentatively proposed for his Bureau. It is a coordinating central office maintained by the British Colonies for strengthening their work on plant diseases. Nearly sixty investigators are included in its field. Some of the lines of work started or shortly to be undertaken are: Correlation of projects, so that each worker may know who is engaged upon a certain problem; collection of literature so as to furnish a worker with a copy, a summary, or a translation of a desired paper which is not available to the person needing it; identification of specimens; pure cultures of parasitic fungi;

and host and parasite indexes. Dr. Butler desires to promote international relations so far as may be possible.

Miss Rose Bracher of the University of Bristol, England, has been spending the past year studying plant pathology in this country. She is the holder of the Rose Sidgwick Memorial Fellowship given by the American Association of University Women, (formerly the Association of Collegiate Alumnae) in memory of Miss Rose Sidgwick of the University of Oxford who died in this country while engaged in work for the British Educational Mission of 1917. Miss Bracher has spent eight months at the University of Wisconsin, one month at Columbia University, two months in the United States Department of Agriculture, and about two weeks at Guelph, Ontario. She returns to England to take up investigations in plant pathology and in the cytology of Rhytisma.

Dr. A. de Jaczewski of the Forestry Institute at Petrograd is visiting various plant pathological centers in this country. He is endeavoring to reestablish exchange relations with other pathologists and to learn what progress has been made by the outside world during the past six years of his isolation by reason of the war.

H. R. Rosen, Associate Professor of Plant Pathology, University of Arkansas, has been granted a year's leave of absence and will leave Fayetteville in September for the Missouri Botanical Gardens, where he will complete his work on the bacterial disease of foxtail and other grasses.

Dr. Alfred H. W. Povah, formerly Assistant Professor of Forest Botany and Pathology at the State College of Forestry, Syracuse, N. Y., now holds the position of Associate Professor of Plant Pathology and Associate Pathologist in the Alabama Polytechnic Institute at Auburn.

Dr. H. H. Whetzel, head of the department of plant pathology at Cornell University, is taking sabbatical leave for 1921–22 in Bermuda. While there he is associated with the department of agriculture of the Bermuda Islands in developing their plant disease survey and research work. He also acts as pathological inspector for exported plants and plant products.

Dr. L. M. Massey is acting head of the department of plant pathology at Cornell in Dr. Whetzel's absence on sabbatical leave.

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